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DIVISION OF LUNG DISEASES

NATIONAL HEART, LUNG, AND BLOOD INSTITUTE

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PROGRAM REPORT: FISCAL 1984

National Institutes of Health
Public Health Service
U.S. Department of Health and Human Services

January 1985

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Foreword

This annual program report provides an overview of basic and clinical research, and the research related to prevention of respiratory diseases which was supported by the NHLBI Division of Lung Diseases during Fiscal Year 1984 (October 1, 1983 through September 30, 1984). The Division's research training and career development activities in the pulmonary field are also summarized in this document.

Further information about any of the Division's programs is readily available. The names, address, and telephone numbers of our staff are given in the organizational chart on page 3. I invite you to contact any staff member or me with your inquiries, requests, and suggestions.

Suzanne S. Hurd

Suzanne S. Hurd, Ph.D.
Director,
Division of Lung Diseases
National Heart, Lung, and Blood Institute

January 1985

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I. INTRODUCTION

The Division of Lung Diseases of the National Heart, Lung, and Blood Institute serves as a focal point within the Institute for planning and coordinating research and research training programs for all diseases of the respiratory system with the exception of lung cancer and infectious diseases of the lung, which are within the purview of the National Cancer Institute and the National Institute of Allergy and Infectious Diseases, respectively. The programs of the Division of Lung Diseases, which are identified in accordance with the requirements of the National Heart, Blood Vessel, Lung, and Blood Act of 1972 (P.L. 92-423), are included in eight major categories. These are listed and described in Section III of this report.

The Division has always committed a major portion of its resources to investigator-initiated research grants and training programs, but it has also attempted to create incentives for exploration of new areas through conferences, workshops, and special initiatives. Such activities for Fiscal 1984 are described in Section IV.

The past dozen years has seen numerous research accomplishments which have had an impact on the effective treatment and prevention of respiratory diseases. Section V contains highlights of some of these scientific advances; they are based on reports submitted to the Division by its grantees and contractors. This section is not intended to be all-inclusive, nor is it intended to suggest that the work described is the most important. Nevertheless, it is hoped that this material will be of interest and will help stimulate further advances.

The last part of this report contains two appendices (A and B). Appendix A lists all of the grants and contracts supported by the Division in Fiscal 1984. Appendix B provides information for those who wish to become more familiar with research and training mechanisms available through the Division of Lung Diseases, the NHLBI, and other units of the NIH.

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II. DIVISION OF LUNG DISEASES ORGANIZATIONAL CHART

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STRUCTURE & FUNCTION BRANCH
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D. Gail, Ph.D., Branch Chief

E. Sinnett, Ph.D.

P. Thadani, Ph.D.

J. Kiley, Ph.D.

Programs
(Grant, Contract, SCOR)

Respiratory Function

Nonrespiratory Function

Lung Cell Biology

Growth & Development (including Neonatal RDS)

AIRWAYS DISEASES BRANCH
(301) 496-7332

S. Ram, Ph.D., Branch Chief

H. Peavy, M.D.

Z. Bengali, Ph.D.

Programs
(Grant, Contract, SCOR)

Asthma

Chronic Obstructive Pulmonary Diseases

Bronchiolitis

Cystic Fibrosis

INTERSTITIAL LUNG DISEASES BRANCH
(301) 496-7034

C. Vreim, Ph.D., Branch Chief

A. Small, Ph.D.

A. Kalica, Ph.D.

Programs
(Grant, Contract, SCOR)

Occupational and Immunologic Lung Diseases

Pulmonary Vascular Diseases

Respiratory Failure

PREVENTION, EDUCATION, AND RESEARCH TRAINING BRANCH
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S. Parker, Ph.D., Branch Chief

J. Kusek, Ph.D.

J. Wolle, Ph.D.

D. Aiken, M.A.

Programs
(Grant, Contract, SCOR)

Prevention, Control, and Education

Research Training Development

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III. NATIONAL PLAN OF THE NHLBI LUNG PROGRAM

The research supported by the NHLBI Division of Lung Diseases is in consonance with the National Plan of the NHLBI Lung Program. Listed below are the Division's National Plan Categories. In the subsections that follow, each Category is described more fully.

A. STRUCTURE AND FUNCTION OF THE LUNG
B. PEDIATRIC PULMONARY DISEASES
C. CHRONIC OBSTRUCTIVE PULMONARY DISEASES
D. OCCUPATIONAL AND IMMUNOLOGIC LUNG DISEASES
E. PULMONARY VASCULAR DISEASES
F. RESPIRATORY FAILURE
G. PREVENTION, CONTROL, AND EDUCATION
H. RESEARCH TRAINING AND CAREER DEVELOPMENT

A. STRUCTURE AND FUNCTION OF THE LUNG

1. Introduction

Described in this subsection are areas of research that deal with the structure and function of the normal developing and adult lung, and the alterations associated with disease. These investigations provide the foundation for understanding the etiology, pathogenesis, and clinical manifestations of respiratory diseases.

2. Long-Range Objectives

Lung Cell Biology

- Characterization of structural and functional features of various types of lung cells, interrelationships among different cell types, and modifications associated with lung injury and disease.

Ventilatory Function

- Improved understanding of gas exchange and transport from the environment to the cell, of utilization at cellular and subcellular levels, and of alterations associated with various stresses such as exercise, high altitude, hyperbaria, and endogenous disturbances.

- Elucidation of respiratory macro- and micromechanics in normal and disease states and of respiratory and circulatory interactions.

- Improved understanding of the roles of chemical, mechanical, and neural factors in control of normal

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ventilation, and in selected states and situations such as sleep, exercise and high altitude.

Nonventilatory Lung Function

- Elucidation of the metabolic activities of the lung, including the surfactant system, and the defense functions of the lung against exogenous insults and endogenous disturbances.

Growth and Development of the Lung

- Understanding of the ventilatory and non-ventilatory functions of the immature respiratory system and their response to endogenous and exogenous insults.

B. PEDIATRIC PULMONARY DISEASES

1. Introduction

Neonatal respiratory distress syndrome, bronchopulmonary dysplasia, cystic fibrosis, and bronchiolitis - the diseases addressed in this subsection - differ markedly in etiology and clinical manifestations. They are, however, linked because they are disorders of infants or young children in which pulmonary involvement is the sole or major cause of morbidity or mortality.

2. Long-Range Objectives

Neonatal Respiration Distress Syndrome

- Improved detection, management, and prevention of neonatal respiratory disorders and their sequelae.

Cystic Fibrosis

- Identification of the basic defect in cystic fibrosis leading to pulmonary disease; characterization of the early pathogenic changes through study of structural and functional derangements in the lung, and improved management of cystic fibrosis through critical assessment of current modes of therapy and development of new regimens.

Bronchiolitis

- Increased understanding of the pathophysiologic features of bronchiolitis and the relationship between bronchiolitis in childhood and subsequent disorders of the respiratory system.

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C. CHRONIC OBSTRUCTIVE PULMONARY DISEASES

1. Introduction

Chronic bronchitis and emphysema receive the major emphasis in this National Plan Category. These are progressive, debilitating disorders that are irreversible by the time they are diagnosed; treatment is only palliative at present. Hence, the primary research focus is on prevention through understanding risk factors and pathogenetic mechanisms, and through arresting disease progression. Asthma, the other chronic obstructive pulmonary disease covered in this Category, is being studied mainly with regard to mechanisms involved in bronchoconstriction, and development of therapeutic interventions.

2. Long-Range Objectives

Chronic Obstructive Pulmonary Diseases

- Elucidation of basic mechanisms involved in structural and functional derangements associated with the onset and progression of chronic bronchitis and emphysema.

- Improved management of chronic obstructive pulmonary diseases through identification of presymptomatic stages, critical assessment of current therapeutic measures, and development of more effective regimens.

- Prevention of chronic obstructive pulmonary diseases through understanding of individual risk factors, and of their interactions and roles in the etiology and pathogenesis of these disorders.

Asthma

- Elucidation of underlying mechanisms in bronchoconstriction and development of more effective measures to ameliorate or prevent the bronchoconstrictor response.

D. OCCUPATIONAL AND IMMUNOLOGIC LUNG DISEASES

1. Introduction

The diseases in this program area comprise a heterogeneous group which are characterized by extensive changes in alveolar supporting tissues. In most of these diseases, the "end-stage" change is pulmonary fibrosis. However, many types of inflammatory reactions occur in the course of the disease process; in some the formation of granulomas predominate. Understanding the involvement of immunologic responses

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in the etiology and pathogenesis of these diseases is an important objective of this program. Early diagnosis, more effective management, and prevention of these interstitial lung diseases are the ultimate goals of the research being carried out in this area.

2. Long-Range Objectives

- Elucidation of the role of immunologic and other basic mechanisms in the onset of interstitial lung diseases and the progressive changes that lead to pulmonary fibrosis and granulomatous lesions.

- Prevention and improved management of these diseases through identification of etiologic agents and other risk factors, early detection of diseases, and development and improvement of therapeutic regimes.

E. PULMONARY VASCULAR DISEASES

1. Introduction

Research related to the pathogenesis of pulmonary edema, hypertension and thromboembolism, and disorders of the pulmonary vasculature is being carried out under the aegis of this National Plan Category. These problems are difficult to diagnose and treat because they are often secondary to other diseases and because clinical symptoms do not appear in the early stages of dysfunction. Of particular interest are endogenous substances that mediate alterations in the lung.

2. Long-Range Objectives

- Elucidation of the mechanisms controlling fluid and solute exchange and hemodynamics in the normal and diseased pulmonary circulation.

- Improve the management of pulmonary vascular diseases through the development of noninvasive diagnostic techniques and more effective therapeutic regimens.

F. RESPIRATORY FAILURE

1. Introduction

Respiratory failure is a frequently fatal condition in which damage to the lung is so severe that gas exchange between alveoli and blood is insufficient to meet the metabolic requirements of the body, and carbon dioxide levels in the blood become excessive. Of immediate concern, because of the high mortality associated with respiratory failure, is better

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management through earlier recognition of impending failure, more accurate diagnosis, and development of more effective therapies. The major research focus is on fundamental investigations to determine the causes of lung injury, identification of progressive changes that result in respiratory failure, and on ways to prevent or reverse this progression.

2. Long-Range Objectives

- Elucidation of intrinsic injury mechanisms which lead to respiratory failure.
- Improved management of acute respiratory failure through development of measures which detect, arrest, or reverse degenerative changes following lung injury.

G. PREVENTION, EDUCATION, AND CONTROL

1. Introduction

The prevention and control program of the Division is concerned with developing education and demonstration programs that will affect the health-related behavior of the constituencies to which they are addressed. Aspects of this problem that are gaining more attention are the roles of social and psychological factors on health-related behavior, and of the effect of established behavioral patterns on the capacity and willingness of individuals to utilize what they learn.

2. Long-Range Objectives

- Develop research, education, and demonstration programs in prevention and control of lung diseases.
- Identify the natural history and personal risk factors affecting the onset, severity, and prevalence of lung diseases in order to control more effectively the course of disease in the population at risk.
- Facilitate the transfer of knowledge gained from biomedical and behavioral research, into the practice of clinical medicine.

H. RESEARCH TRAINING AND CAREER DEVELOPMENT

1. Introduction

Programs for development of manpower for pulmonary research and academic medicine are supported through both NIH-wide mechanisms and special initiatives of the Division of Lung Diseases. In the former category are National Research Service

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Awards (NRSAs) for institutions and individuals, and Research Career Development Awards (RCDAs). A special Division initiative is the NRSA Minority Summer Program for Pulmonary Research which was announced in Fiscal 1984 for implementation in Fiscal 1985. In addition, the Division participates in special NHLBI initiatives, namely, the Clinical Investigator Award and the Physician Scientist Award.

2. Long-Range Objectives

- To provide training for research related to the respiratory system and academic careers in pulmonary medicine.

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IV. ACTIVITIES FOSTERED IN FISCAL 1984

The investigator-initiated research grant continues to be the major source of innovative fundamental research. It contributes not only to our understanding of normal lung function and the processes involved in respiratory disorders, but also leads to improvements in the diagnosis and management of pulmonary diseases. From time to time, the Division supplements these programs by issuing requests for applications (RFAs) or for contract proposals (RFPs) to encourage exploration of problems not being addressed, or being insufficiently addressed, by investigator-initiated grants. The areas where stimulation is needed are identified through working groups, workshops, and task forces, as well as by the Pulmonary Diseases Advisory Committee and the Division's professional staff. Below is a synopsis of the Division's activities during Fiscal 1984 aimed at achieving the goals of the National Plan of the NHLBI Lung Program.

A multicenter, multidisciplinary clinical study entitled Prospective Investigation of Pulmonary Embolism Diagnosis was implemented in September 1983. The purpose of this study is to determine the sensitivity and specificity of ventilation-perfusion scans as compared to angiograms for the diagnosis of acute pulmonary embolism. Contracts were awarded to six clinical centers and a data and coordinating center. The protocol has been completed and approved. The manual of operations and the development and pretesting of patient reporting forms were completed in December 1984. Patient recruitment began in January of 1985, and will continue for two years. Scans and angiograms will be obtained in a total of 900 patients. The clinical phase of this study will end in September 1987.

As of June 1984, 183 patients had been enrolled in the Primary Pulmonary Hypertension Registry. About 60% of the patients were female, with the majority being in the age range of 20-39 years. Mortality data have been obtained on all patients and show that approximately 32% of the patients enrolled in the registry are no longer alive. Patient enrollment will continue through September 1985 and data analysis will be completed in March 1986.

A clinical trial on High Frequency Ventilation in Premature Infants began in August 1984, with an initial eight-month planning phase devoted to protocol development. The purpose of the trial is to evaluate the efficacy and possible risks of high frequency ventilation in premature infants. A second clinical trial, Early Intervention for Chronic Obstructive Pulmonary Diseases, was also initiated in August 1984. Its purpose is to determine the rate of decline in pulmonary function during "special care," as compared with usual care, in cigarette smokers with mild abnormalities in pulmonary function. Development of the specific protocol should be completed by September 1985.

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A request for applications (RFA) entitled The Genetic and Metabolic Defects Underlying Cystic Fibrosis was issued jointly by the Division and the National Institute of Arthritis, Diabetes, and Digestive and Kidney Diseases. Its purpose is to better define and characterize the basic metabolic defects associated with the etiology and pathogenesis of cystic fibrosis. Seven applications received in response to the RFA were assigned to the Division, and two were funded. The Division also received five applications in response to the RFA on Demonstration and Education Research issued by the NHLBI; it funded two of these applications.

Three other solicitations, all RFAs, were announced by the Division this past year: Production of Toxic Oxygen Species in Acute Lung Injury, which attracted eighteen applications with five funded, Isolation and Characterization of Human Lung Cells, which resulted in twenty-two applications with six funded, and Markers for Endothelial Injury and Repair, for which thirteen applications were received and six funded.

The Division sponsored several workshops and meetings during Fiscal 1984. A workshop on the Pulmonary Complications of the Acquired Immune Deficiency Syndrome (AIDS) held in October 1983 was designed to focus attention on the unique problems that face pulmonary physicians who treat patients with AIDS. The specific aims of the workshop were: 1) to review current knowledge of the pulmonary complications of AIDS; 2) to review, consolidate, and summarize the experiences and practices from hospitals in the United States in which considerable numbers of AIDS patients are treated; and 3) to recommend areas to further research related to this problem. The proceedings of the workshop have been published in the New England Journal of Medicine 310:1682-1688 (June 21), 1984 and is also available as a summary report from the Division of Lung Diseases.

An International Conference on Bronchoalveolar Lavage (BAL) was held at Columbia, Maryland, in May 1984. Three hundred scientists, of whom about one hundred were from foreign countries, attended the conference. Topics considered were the methodology of BAL, the influence of cigarette smoking on BAL results, the use of BAL in asthma patients, BAL as applied to patients with interstitial lung diseases, the use of BAL to assess acute lung injury, and the role of BAL in occupational lung diseases. A program, containing the agenda and 156 abstracts, is available from the Division. A summary of the meeting is now being prepared for publication.

Mechanisms of Inflammation in Asthma was the title of a workshop held in February 1984. Thirty-three investigators represented the fields of pulmonary medicine, allergy, immunology, pharmacology, pathology, physiology, and biochemistry. A report is being prepared for dissemination to the scientific community.

A workshop on Clinical Use of Lung Water Measurements was held to review and discuss the use of lung water measurements for studying the mechanisms of pathogenesis and resolution of pulmonary edema,

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and for diagnosing and managing patients with this disorder. Current radiologic, nuclear, and indicator dilution techniques for measuring lung water were also presented. A report on the workshop is in preparation.

The workshop on Cell-to-Cell Interaction in the Developing Lung brought together investigators participating in the RFA program of the same name with pulmonary researchers and cell biologists studying cell communication in other organs including the eye and developing nervous system. The meeting emphasized the need for future studies of basic biology of the developing lung. A report has recently been published. (Tissue and Cell 16:829-842, 1985).

Two workshops were held in September 1984. The focus of the workshop entitled Respiratory Sensations was on the sensation of dyspnea. Speakers addressed the topic from several perspectives, which included proprioception, psychophysical techniques, physiology, and mechanisms and interventions. A summary of the workshop will be published in the Journal of Applied Physiology. The main thrust of the workshop on Diagnosis of Emphysema was to define criteria to aid in diagnosis of emphysema and evaluation of pathologic material. A paper resulting from this workshop will be submitted for publication in the near future.

An Occupational and Immunologic Lung Diseases SCOR Coordination Meeting was held to discuss plans for a proposed collaborative effort aimed at standardizing BAL procedures and evaluating the use of BAL in diagnosing, staging, and managing idiopathic pulmonary fibrosis patients as compared to patients with other selected interstitial lung diseases, namely, connective tissue disorders, and disorders due to exposure to silica and asbestos. In line with the Division's Occupational and Immunologic Lung Diseases Program, a working group on Prevention of Occupational Lung Diseases met to discuss methods of encouraging research into prevention of such diseases. The participants agreed that demonstration and education research relevant to occupational lung diseases should be stimulated.

A conference on Aspiration Hazards to the Developing Lung was sponsored by the American Thoracic Society and supported by a grant from the Division. The conference included in-depth, critical evaluation of the ontology of swallowing, esophageal motility, gastroesophageal function and mechanisms to protect the integrity of the airways. The proceedings will be published in the American Review of Respiratory Disease and will help form the basis for improved approach to diagnosis, and for treatment and prevention of foreign body and other aspiration syndromes in childhood.

The Division collaborated with the National Institute of Child Health and Human Development to co-sponsor the First Annual Meeting of the Fetal Medicine and Surgery Society which was held in Washington, D.C. The focus of the meeting was to discuss the state of the art of diagnosing and correcting structural defects and

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functional abnormalities related to pulmonary, renal, neurologic, and metabolic disorders in the fetus in utero.

During Fiscal 1984 the following publications emanated from activities sponsored by the Division of Lung Diseases. Copies are available from the Division.

- o Intermittent Positive Pressure Breathing Therapy of Chronic Obstructive Pulmonary Diseases. Annals of Internal Medicine, 99:612-620, 1983.

- o Report of workshop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies held May 1-3, 1983. Issued December 1983 by the NHLBI Division of Lung Diseases.

- o Report of workshop on Exercise Testing in the Dyspneic Patient held September 1-2, 1982 in Bethesda, Maryland. This report appeared as a supplement to Volume 129 of the American Review of Respiratory Disease published in February 1984.

- o Effects of Antenatal Dexamethasone Administration in the Infant: Long-Term Follow-Up. The Journal of Pediatrics, 104:259-267, February 1984.

- o Report of workshop on Pulmonary Complications of the Acquired Immunodeficiency Syndrome (AIDS) held October 13-14, 1983, in Bethesda, Maryland. This report was published by the Division in July 1984. Attached to it is a Special Report on the workshop which appeared in The New England Journal of Medicine 310:1682-1688 (June 21), 1984.

- o Report of workshop on The Fibroblast in Interstitial Lung Diseases held September 14-15, 1982, in Bethesda, Maryland. This report became available in August 1984.

During the final weeks of the fiscal year, two requests for applications (RFAs) were prepared inviting new and renewal applications for four of the Division's Specialized Center of Research (SCOR) programs. The first RFA (receipt date September 2, 1985) requests applications concerned with respiratory disorders of neonates and children. The second (receipt date December 2, 1985) requests applications in chronic airways diseases, occupational and immunologic lung diseases, or pulmonary vascular diseases. Both RFAs also request applications for National Research and Demonstration Centers (NRDCs). The aim of this new grant instrument is to enhance the effectiveness of a traditional specialized center grant through demonstration and education research. Copies of both RFAs are available from the Division of Lung Diseases.

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V. HIGHLIGHTS OF SCIENTIFIC ADVANCES

The aim of this section is to inform the biomedical community about some of the research programs supported by the Division of Lung Diseases. This material is based on annual reports submitted by grantees and contractors. It should be recognized that many other exciting and important research findings could not be included in this brief summary.

Chronic Obstructive Pulmonary Disease The Surgeon General's 1984 Report on the Health Consequences of Smoking, which this year dealt with Chronic Obstructive Pulmonary Disease (COPD), highlights the rapid rise in the number of deaths from COPD over the past fourteen years. In 1983, COPD related to smoking accounted for almost 62,000 deaths, compared to 33,000 deaths in 1970. Between 1970 and 1980, the last year for which complete mortality data are available, a significant shift was seen between the male to female ratio of these deaths. In 1970, male deaths outnumbered female deaths by a ratio of 4.3 to 1. By 1980, this ratio had declined to 2.36. This shift is seen as the result of the rapid rise in cigarette smoking among women since World War II.

Cigarette smoking also is the major cause of COPD morbidity in the United States; 80 to 90 per cent of the COPD in the United States is attributable to it. In population-based studies over the past fifteen years, cigarette smoking is often the only significant predictor for the development of COPD. Estimates of the prevalence of airway obstruction range from one population to another, but for men, estimates range from 3.6% to 5.6%. For women the estimates of prevalence range from 1.9% to 3.4% of the population. It is expected that morbidity and mortality from COPD will continue to rise in the coming years. In 1980, men who smoke comprised 36.8% of the male population, and women smokers 29.3% of the female population. Unless these trends are reversed, illness and death from this disease will continue to rise.

Passive Smoking Children whose parents smoke have significantly more respiratory illness in the first year of life. Detrimental effects of passive smoking on the lung function of exposed children have been more difficult to document than the incidence of infection; it is believed that both factors may contribute to the future development of airways obstruction. Recently, it was concluded from the first longitudinal study on the effects of parental smoking that children whose mothers smoke have a seven per cent lag in lung growth compared to children of nonsmoking mothers. In predicting the effect of passive smoking on a child's lung function, the mother's smoking status was found to be the most important factor. Results from two additional population studies have now confirmed the finding of diminished lung function in children whose mothers smoke. The medical significance of these early respiratory effects of passive smoking remain unknown, but to the extent that small decreases in pulmonary function persist, they

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may represent an important predictor of future respiratory problems, especially among those children who themselves become cigarette smokers. Long-term follow-up of children from homes where parents smoke and comparisons of data obtained by different research groups will be necessary before this potentially important health issue can be resolved. In the meantime, parents - especially mothers - can eliminate this possible risk to their children by not smoking.

One of the problems encountered in conducting longitudinal studies on the effects of passive smoking is that only rough estimates of the individual's exposure to smoke have been possible. These have been based on questionnaires regarding the number of smokers in the household. However, a more precise measure is now available. Researchers who are investigating the effect of passive smoking in children have developed a method which will reliably measure the exposure of infants to second-hand cigarette smoke by measuring the amount of nicotine metabolites in infants' urine. A pilot study, recently published in the New England Journal of Medicine showed that infants in households that include smokers absorb tobacco smoke constituents and excrete nicotine in their urine, in addition to metabolizing nicotine to form cotinine. Infants in smoking households showed significantly higher concentrations of nicotine and cotinine than infants not exposed to household smoke. Moreover, the greater the number of cigarettes the mother reported smoking, the higher the concentration of nicotine and cotinine in the infant's urine. In fact, some of the infants showed urinary and salivary concentrations of nicotine and cotinine that are within the range found in light smokers.

This study is important for two reasons. First, it has shown that infants exposed to second-hand tobacco smoke actually absorb it. Secondly, a method is now available to provide an objective measure of the amount of smoke absorbed by the infant, which can be used to study the short and long-term health effects of passive smoking.

Asthma Self-management The National Heart, Lung, and Blood Institute, the American Lung Association, and Boehringer-Ingelheim have developed a joint program to disseminate pediatric asthma self-management educational materials to health care professionals, asthmatic children, and their parents. Boehringer-Ingelheim has provided a grant to the American Lung Association to support four regional workshops to teach health care professionals how to implement asthma self-management programs developed and evaluated by the NHLBI, and to disseminate a self-help package ("Superstuff") developed by the American Lung Association. In addition, the drug company has provided funds to replicate each of the self-management programs in each of the four regions. The NHLBI is also preparing a workshop planning package to assist 145 additional local ALA groups and other voluntary organizations who wish to hold workshops of their own for local health care professionals.

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The three asthma self-management programs developed by the NHLBI showed successful results. Reductions in hospitalizations, emergency room visits, and days lost from school were evidence of the greater responsibility children and parents took to manage the asthma. The widespread use of these programs can have a valuable positive effect on the health of the two million asthmatic children in this country.

High Frequency Ventilation Currently investigations are providing the basis for developing new therapies for premature infants with pulmonary complications. It is currently estimated that, on average, 20 per cent of infants below 2000 g who are supported by mechanical ventilation proceed to develop chronic lung disorders. Two factors considered to be important in the pathogenesis of these disorders are barotrauma, which is caused by the use of high pressures in standard mechanical ventilators, and oxygen toxicity, which is caused by the use of high levels of oxygen for extended periods. Unfortunately, using high pressures and high concentrations of oxygen is often the only way that doctors have had to keep some premature babies alive during the early postnatal period. Recent animal and human studies now suggest that adequate oxygenation and carbon dioxide removal can be provided by a new device that provides what is called high frequency ventilation (HFV), using less inspired oxygen and lower airway pressures than are required with standard ventilation.

During Fiscal 1984, the Division initiated a clinical trial to evaluate whether HFV is superior to standard mechanical ventilation in providing ventilatory support to premature infants. Roughly 1,000 infants will be enrolled at ten hospitals beginning in the Spring of 1985. Enrollment is expected to continue for one year and will be followed by a second twelve month period to allow for the testing of neurological and respiratory development when each infant is one year old. The results of the trial will show whether HFV can significantly reduce both the incidence of chronic lung diseases in premature infants and the period over which mechanical ventilation must be employed. Whatever the results, the outcome of this study should have significant effects on the methods used to ventilate premature infants.

Hormones in Fetal Lungs Another interesting possibility for helping premature infants with pulmonary complications comes from studies concerning the interaction of two important hormones, corticosteroids and thyroid hormones, in fetal lung. Previous studies on lung tissue *in vitro* have shown that a thyroid hormone, triiodothyronine (T_3), enhances the effects of dexamethasone in stimulating synthesis of a key component of lung surfactant called phosphatidylcholine (PC). A recent study examined the effects of T_3 and a corticosteroid, betamethasone, with regard to PC synthesis by fetal rat lung *in vivo*. The results showed that injections of either hormone caused a stimulation of PC synthesis by approximately 33%, and that

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administration of both hormones together produced a 69% increase. These data suggest that there is an additive interaction between T_3 and betamethasone with regard to synthesis of surfactant lipid. This supports the view that combined hormone administration may be more effective than single hormones in preventing neonatal respiratory distress syndrome, a disease characterized by a deficiency of surfactant. More information is clearly required before clinical use of combined hormone therapy can be considered.

Adult Respiratory Distress Syndrome Patients with sepsis are at high risk of developing acute lung injury, which can lead to pulmonary edema and finally to adult respiratory distress syndrome (ARDS). It has been suggested that complement activation plays an important role in causing acute lung injury in patients with sepsis and that complement activation may be used in predicting the development of ARDS in patients at risk. Four indices of complement activity - neutrophil chemotaxis, neutrophil aggregation, plasma concentrations of C3a des Arg, and of C5a des Arg - were recently studied in forty patients with the sepsis syndrome. Acute lung injury was assessed by a composite scoring system that took into account chest x-ray abnormalities, presence and severity of hypoxia, and respiratory system compliance. Nearly all patients had elevated levels of C3a des Arg and C5a des Arg when first seen. However, neither of these tests nor those of neutrophil chemotaxis or neutrophil aggregation correlated with the presence of acute lung disease; and none of the indices were able to predict which patients would develop lung injury. From this study it has been concluded that biologic and immunologic assays of intravascular complement activation, which are often positive in patients with sepsis, neither correlate with the initial severity nor predict the development or worsening of associated lung injury. The results of this study decreases the likelihood that complement activation plays a major role in the pathogenesis of acute lung injury in septic patients. Also, currently available immunologic and biologic assays of complement activation appear not to be of clinical value in the identification of septic patients who present with or develop progressive acute lung injury. These findings could have important implications in the design of effective therapy for the prevention and treatment of adult respiratory distress syndrome in septic patients.

Oxygen Toxicity High concentrations of oxygen are toxic to the lungs of all species, including man. The principal biochemical defense against oxygen-induced lung damage is generally accepted to be the antioxidant enzymes which include superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GP). Previous work has established that the relative resistance to hyperoxia appears to be correlated with increased levels of some or all of these pulmonary antioxidant enzymes. It has also been noted that activities of some of these enzymes are higher in the lungs of newborn animals than in the lungs of premature fetuses. A recent study has examined the activities of these enzymes in developing lungs of rabbit fetuses at specific intervals during gestation and continuing through several days after

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birth. The aim of the study was to determine if some specific changes occur in the developing lung to help assure its successful adaptation from the utering environment, which is relatively low in oxygen, to a comparatively hyperoxic environment after birth. The results showed that SOD, CAT, and GP activities exhibited similar maturational patterns with significant increases in activity during the last three to five days before birth, as compared with earlier gestational levels. For example, during the final days in utero, SOD and CAT activities increased by 110%, and lung GP activity by 200%.

These data are consistent with work of others, and suggest that the antioxidant system of developing lungs shows a rapid rise in activity late in gestation - as is the case with the surfactant system. These prenatal increases in biochemical activity appear to be important preparations for birth that will allow the newborn's lung to assume effective gas exchange functions. The prematurely born may therefore be handicapped in its ability to tolerate hyperoxic exposure and to resist serious oxygen-induced lung pathology. These data may explain the clinical observation that prematurely born infants seem to be more susceptible to developing bronchopulmonary dysplasia after oxygen therapy.

Elastin Synthesis Tissues consist of cells and a supporting extracellular matrix which provides a scaffolding for the tissue. The matrix is a highly complex organized system of macromolecules and, like the cell populations, its composition and structure are unique to each tissue and suited to the specific functions of the organ of which it is a part. The basic components of the matrix are synthesized by the cells which it surrounds. An im- portant component of the pulmonary extracellular matrix is elastin which gives the lung elastic recoil. Large scale destruction of elastin (for example, due to cigarette smoking) results in the breakdown of the pulmonary tissue. Researchers have found important clues towards understanding the synthesis of elastin by cells. Elastin synthesis occurs at a rather late stage during development and appears to be influenced by the extracellular matrix. Undifferentiated (nonelastin producing) cells from animals of early gestational ages were found to synthesize elastin when grown on extracellular matrix prepared from ligaments of animals of late gestational ages. Extracellular matrix from animal fetuses which are too young to produce elastin does not stimulate elastin production in undifferentiated cells. Researchers have now found special receptors for elastin on surfaces of cells which have "learned" to synthesize elastin. These receptors are not present on very young, undifferentiated cells. Further understanding of the nature and properties of these receptors and mode of their operation would throw light upon the repair processes in operation in the lung.

Surfactant in Acute Lung Injury In patients with the Adult Respiratory Distress Syndrome (ARDS), a form of acute respiratory failure associated with a high mortality rate, the lungs are unable to effectively transfer oxygen from the air spaces to the blood in spite of being supplied with a high concentration of

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oxygen. One of the reasons that oxygen transfer fails is the collapse of the smallest air spaces (alveoli) of the lungs, a process known as atelectasis. During the course of this disease, the predominant lining cells of the alveoli, known as type I cells, are damaged, and, as healing proceeds, they are replaced with type II cells, which normally make up a small fraction of the alveolar lining. The type II cells are also the source of lung surfactant, a material which is responsible for reducing surface tension in the alveoli and preventing their collapse. For this reason, investigators studying ARDS have long been puzzled by the persistent atelectasis seen in this disorder. Certainly, it would seem, the large number of type II cells should provide more than enough surfactant to keep the alveoli open. Recent studies in baboons with experimentally-induced lung injury have now provided a possible explanation for this situation. After the number of type II cells had greatly increased, surfactant material was recovered by washing the lungs with a salt solution. Although, as expected, there was an increase in the total amount of lipids and proteins, the substances that constitute the pulmonary surfactants, they were abnormal in their chemical composition. When the surfactant material was tested for its ability to lower surface tension, its activity was found to be markedly reduced. Since reversal of the atelectasis which characterizes ARDS probably depends on the presence of adequate lung surfactant, these studies help to explain why recovery from this disease is delayed.

Pulmonary Fibrosis The mechanisms by which inflammation in the lung progresses to fibrosis is of central importance to understanding pulmonary fibrotic diseases. One of the early events at the site of inflammation is accumulation of cells (macrophages) whose function is to protect the host from injury or infection. By studying the ability of human alveolar macrophages to modulate fibrin following inflammatory stimuli, scientists are beginning to determine mechanisms that appear to be involved in the early events leading to the formation of fibrotic lesions in the lung. Fibrin, an insoluble protein that normally forms the essential portion of a blood clot, has long been recognized to be an important element in the inflammatory process. The rate of fibrin formation and its resorption (digestion) are considered to be important early events in the regulation of scar tissue formation during the healing process. The cells (fibroblasts) that are responsible for production of scar tissue use fibrin as a means of attachment and accelerated growth at the inflammatory site. A correlation has been observed between the amount of fibrin deposited in inflammatory reactions and the subsequent localization of fibrotic processes in the lung.

Recently, it was shown that human alveolar macrophages produce a substance (plasminogen activator) that promotes digestion of fibrin. It was also shown that human alveolar macrophages produce a substance similar in function to factor VII, a substance in blood that participates in fibrin accumulation in blood clots. Thus human alveolar macrophages appear to produce substances which are capable of influencing both formation and digestion of fibrin at inflammatory sites in the lung.

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The possible significance of these findings is seen from results of studies with sarcoidosis patients. Sarcoidosis is a chronic inflammatory disease that may result in pulmonary fibrosis. When alveolar macrophages from sarcoidosis patients were tested, they were observed to have increased factor VII activity. In addition, specially-stained lung tissue samples from these patients showed fibrin accumulations only in these portions that were rich in macrophages. Together, these findings support the view that fibrin accumulation may precede and support the subsequent fibrotic lesions found in the lungs of sarcoidosis patients. In addition, these findings offer the possibility that tissue fibrin may eventually be used as a marker for determining the risk of developing of fibrotic lesions and might provide new strategies for prevention and treatment of fibrotic lung diseases.

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APPENDIX A

DIVISION OF LUNG DISEASES GRANTS AND CONTRACTS SUPPORTED
IN FISCAL 1984

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1. ADAIR, THOMAS H — R01 HL32049-01 — UNIVERSITY OF MISSISSIPPI MEDICAL CTR
   MODIFICATION OF LYMPH BY LUNG AND SYSTEMIC LYMPH NODES

2. ADAMS, GEORGE K — R01 HL30132-02 — JOHNS HOPKINS UNIVERSITY
   CHEMICAL MEDIATORS AND HUMAN AIRWAY ANAPHYLAXIS

3. ADAMS, JAMES M — R01 HL29396-02 — UNIVERSITY OF VIRGINIA CHARLOTTESVILLE
   CARBON DIOXIDE TRANSPORT AND CHEMOSENSITIVITY

4. ADAMS, JAMES M — R01 HL29737-02 — UNIVERSITY OF VIRGINIA CHARLOTTESVILLE
   MEDULLARY CHEMORECEPTOR PHYSIOLOGY

5. ADAMSON, IAN Y — R01 HL28188-03 — UNIVERSITY OF MANITOBA
   ROLE OF INTERCELLULAR CONTACTS IN LUNG DEVELOPMENT

6. AHMED, TAHIR — R23 HL27798-03 — MOUNT SINAI MEDICAL CENTER (MIAMI BEACH
   HISTAMINE RECEPTOR SYSTEM IN BRONCHIAL ASTHMA

7. ALAVI, ABASS — N01 HR34011-02 — PENNSYLVANIA, UNIVERSITY OF
   EVALUATE SCANS AND ANGIOGRAPHY IN PULMONARY EMBOLISM

8. ALBERTINE, KURT H — R23 HL32736-01 — UNIVERSITY OF SOUTH FLORIDA
   SITE OF LEUCOCYTE - MEDIATED MICROEMBOLISM LUNG INJURY

9. ALBRIGHT, JOHN T — R01 HL23338-03 — BOSTON UNIVERSITY
   THE ANTIOXIDANT SYSTEM OF GAS-FILLED ORGANS

10. ALTOSE, MURRY D — N01 HR46020-01 — CASE WESTERN RESERVE UNIVERSITY
    INTERVENTION FOR CHRONIC OBSTRUCTIVE PULMONARY DISEASE

11. ANTHONISEN, NICKOLAS R — N01 HR46017-01 — MANITOBA, UNIVERSITY OF
    EARLY INTERVENTION FOR CHRONIC OBSTRUVTIVE PULMONARY DIS

12. AUST, STEVEN D — R01 HL33543-01 — MICHIGAN STATE UNIVERSITY
    LUNG MICROSOMAL OXYGEN ACTIVATION

13. AUSTEN, K FRANK — P01 HL17382-11 — BRIGHAM AND WOMEN'S HOSPITAL
    CHEMICAL MEDIATORS OF ACUTE PULMONARY DISORDERS

14. AUTOR, ANNE P — R01 HL32082-01 — UNIVERSITY OF BRITISH COLUMBIA
    TOXIC RESPONSE OF LUNG CELLS TO OXYGEN FREE RADICALS

15. AVERY, MARY E — P50 HL27372-02 — BRIGHAM AND WOMEN'S HOSPITAL
    SCOR: PEDIATRIC PULMONARY DISEASE

16. BAENZIGER, NANCY L — R01 HL33699-01 — WASHINGTON UNIVERSITY
    HISTAMINE METABOLISM IN HUMAN PULMONARY VASCULAR CELLS

17. BAILEY, WILLIAM C — N01 HR46019-01 — ALABAMA, UNIVERSITY OF
    INTERVENTION FOR CHRONIC OBSTRUCTIVE PULMONARY DISEASE

18. BAKER, DAVID G — R23 HL27319-03 — UNIVERSITY OF CALIFORNIA SAN FRANCISCO
    CONTROL OF AIRWAY SMOOTH MUSCLE AND MUCOUS GLANDS

19. BALIS, JOHN U — R01 HL29498-03 — UNIVERSITY OF SOUTH FLORIDA
    BIOCHEMICAL MARKERS OF PERINATAL HUMAN SURFACTANT

20. BANCALARI, EDUARDO H — R01 HL25023-04 — UNIVERSITY OF MIAMI
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CYSTIC FIBROSIS: MODULATION OF LUNG MUCUS SECRETION

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MECHANISMS OF PHYSIOLOGICAL RESPONSES TO EXERCISE

602. WATKINS, CLYDE A R01 HL30187-03 MEDICAL COLLEGE OF GEORGIA
EFFECT OF INJURY ON NONRESPIRATORY FUNCTION OF THE LUNG

603. WEBBER, CHARLES L R01 HL32888-01 LOYOLA UNIVERSITY MEDICAL CTR
SYNCHRONIZATION OF PHRENIC MOTONEURONS

604. WEG, JOHN G N01 HR34010-02 MICHIGAN, UNIVERSITY OF
EVALUATE SCANS AND ANGIOGRAPHY IN PULMONARY EMBOLISM

605. WEIL, JOHN V P01 HL14985-13 UNIVERSITY OF COLORADO HLTH SCIENCES CTR
ADAPTATIONS TO HYPOXIA

606. WEILL, HANS P50 HL15092-13 TULANE UNIVERSITY OF LOUISIANA
IMMUNE FIBROTIC RESPONSES TO OCCUPATIONAL ENVIRONMENTS

607. WEINBAUM, GEORGE R01 HL29734-02 GRADUATE HOSPITAL
OXIDANT EFFECTS ON ELASTASE & ALPHA-1-PROTEASE INHIBITOR

608. WEISSMANN, GERALD R01 HL19721-09 NEW YORK UNIVERSITY
LUNG INFLAMMATION: LYSOSOMAL PROTEASES AND LIPOSOMES

609. WEISZ-CARRINGTON, PAUL R01 HL32575-01 VETERANS ADMIN MED CTR,SHREVEPORT,LA
SECRETORY IMMUNITY AFTER ORAL IMMUNIZATION

610. WELSH, MICHAEL J R01 HL29851-02 UNIVERSITY OF IOWA
PATHOPHYSIOLOGY OF RESPIRATORY TRACT FLUID PRODUCTION

611. WEST, JOHN B P01 HL17731-10 UNIVERSITY OF CALIFORNIA SAN DIEGO
PHYSIOLOGICAL BASIS OF PULMONARY DISEASE

612. WEST, JOHN B R01 HL28305-02 UNIVERSITY OF CALIFORNIA SAN DIEGO
PULMONARY CAPILLARY MORPHOLOGY DURING HYPOXIA OR EDEMA

613. WESTENSKOW, DWAYNE R01 HL25553-04A1 UNIVERSITY OF UTAH
BIOINSTRUMENTATION FOR DIFFERENTIAL VENTILATION

614. WHITE, DAVID P R23 HL31994-01 PENNSYLVANIA ST UNIV HERSHEY MED CTR
METABOLIC RATE AND BREATHING DURING SLEEP

615. WHITNEY, PHILIP L R01 HL32109-01 UNIVERSITY OF MIAMI
LUNG DEVELOPMENT: ROLE OF ENDOGENOUS LUNG LECTIN

616. WHITSETT, JEFFREY A R01 HL28623-03 UNIVERSITY OF CINCINNATI
REGULATION OF SURFACTANT RELEASE IN TYPE II LUNG CELL

617. WILLOUGHBY, WILLIAM F R01 HL19819-08 JOHNS HOPKINS UNIVERSITY
CELLULAR MECHANISMS OF INFLAMMATORY LUNG DISEASE

618. WILSON, FRANK J, JR R23 HL28174-03 UNIVERSITY OF ARKANSAS MED SCIS LTL ROCK
RESPIRATORY TRACT DEPOSITION OF SMALL AEROSOLS

619. WILSON, IRWIN B R01 HL22242-07 UNIVERSITY OF COLORADO AT BOULDER
THE CONVERTING ENZYME OF LUNG

620. WINDSOR, RICHARD A R18 HL30630-02 UNIVERSITY OF ALABAMA IN BIRMINGHAM
SMOKING CESSATION PROGRAMS FOR UNIVERSITY EMPLOYEES

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621. WOLIN, MICHAEL S R23 HL31069-01A1 NEW YORK MED COLLEGE
PULMONARY VASODILATION AND GUANYLATE CYCLASE REGULATION

622. WOO, SAVIO L R01 HL27509-03 BAYLOR COLLEGE OF MEDICINE
THE ALPHA-1-ANTITRYPSINGENE AND PULMONARY EMPHYSEMA

623. WOOD, LAWRENCE D R01 HL30835-02 UNIVERSITY OF CHICAGO
TREATMENT OF ACUTE HYPOXEMIC RESPIRATORY FAILURE

624. WRIGHT, ELIZABETH C N01 HR72901-16 GEORGE WASHINGTON UNIVERSITY
INTERMITTENT POSITIVE PRESSURE BREATHING--DATA STUDY

625. WRIGHT, JO RAE R23 HL30923-02 UNIVERSITY OF CALIFORNIA SAN FRANCISCO
ALVEOLAR FACTORS IN UPTAKE OF LUNG SURFACTANT

626. YANKASKAS, JAMES R R01 HL33473-01 UNIVERSITY OF NORTH CAROLINA CHAPEL HILL
TISSUE CULTURE AND RESPIRATORY EPITHELIAL FUNCTION

627. YOSHIDA, TAKESHI R01 HL29382-02 UNIVERSITY OF CONNECTICUT HEALTH CENTER
ANERGY IN PULMONARY GRANULOMES

628. YOUNES, MAGDY K R01 HL32030-01 UNIVERSITY OF MANITOBA
CONTROL OF BREATHING IN HEALTH AND DISEASE

629. YOUNG, STEPHEN L R01 HL32188-01 DUKE UNIVERSITY
FETAL YOUNG BIOCHEMISTRY AND 3-DIMENSIONAL MICROANATOMY

630. ZAPOL, WARREN M P50 HL23591-06 MASSACHUSETTS GENERAL HOSPITAL
PULMONARY SCOR: ADULT RESPIRATORY FAILURE

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APPENDIX B

INFORMATION ABOUT RESEARCH AND TRAINING SUPPORT MECHANISMS

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INFORMATION ABOUT RESEARCH AND TRAINING SUPPORT MECHANISMS

Investigator-initiated grants are the major instruments used to fund research related to the various programs of the NHLBI's Division of Lung Diseases. The traditional Research Project Grant (R01) is by far the most common. It supports a discrete project carried out by the named investigator(s) in their area of expertise. Linked rather closely to the traditional research grant is the New Investigator Research Award (R23). The Division also supports Conference (R13) and Demonstration and Education Research (R18) Grants through investigator-initiated submissions. (The latter may also be submitted in response to an NHLBI-issued announcement.)

Multi-project grants, namely, the Research Program Project Grant (P01) and the Specialized Center Grant (P50) are funded by the Division. A program project generally involves the organized efforts of a relatively large number of investigators who conduct projects with a central research focus or objective. The key feature of a Specialized Center of Research is its multidisciplinary attack on a specific pulmonary disease, with clinical focus as a requirement. Specialized centers differ from program projects in that they are developed in response to a request for applications from the Division. Closely linked to specialized centers is a new grant instrument which was recently announced (See page 14). It is the National Research and Demonstration Center Grant (P60), aimed at enhancing traditional specialized centers grants through the addition of demonstration and education research.

The Division's manpower development programs are mainly supported by grants. These are:

(1) the Institutional National Research Service Award (T32), which provides support for predoctoral and postdoctoral research training,

(2) the Postdoctoral Individual National Research Service Award (F32),

(3) the National Research Service Award for Senior Fellows (F33),

(4) the NRSA Short-Term Training Grant (T35),

(5) the NRSA Minority Summer Program for Pulmonary Research (T35S),

(6) the Research Career Development Award (K04),

(7) the Clinical Investigator Award (K08), and

(8) the Physician Scientist Award (K11).

Planned for implementation in Fiscal 1985 is the Minority Institutional Research Training Grant and the Minority School Faculty Development Award.

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The Division promotes targeted research and training through issuance of requests for grant applications (RFAs) and requests for contract proposals (RFPs). Solicitations for these specific programs are formally announced in the NIH Guide for Grants and Contracts, which is published about ten times a year. Investigators who do not have access to this publication through their library or business office can have their institution placed on the mailing list by writing to the Grants and Contracts Guide Distribution Center, National Institutes of Health, Building 31, Room B3N10, Bethesda, Maryland 20205.

Prospective and current grantees are encouraged to contact one of the Division program administrators listed on page 3 for more information about any of the support mechanisms identified above or about any of the scientific programs listed on page 3 or described elsewhere in this report. Program administrators may be contacted by letter addressed to them at 6A16 Westwood Building, NHLBI Division of Lung Diseases, Bethesda, Maryland 20205. Alternately, these individuals may be reached at the telephone numbers for the various units of the Division which are listed in the Organizational Chart on page 3.

Representative literature about extramural programs of other components of the National Heart, Lung, and Blood Institute and of the National Institutes of Health is listed below. These publications are available on request from the Grants Inquiries Office, 449 Westwood Building, NIH Division of Research Grants, Bethesda, Maryland 20205.

- NIH Extramural Programs, NIH Publication No. 83-33
- Profiles of Financial Assistance Programs, DHHS Publication No. 83-50,002
- 1984 NIH Almanac, NIH Publication No. 84-5

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